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Submassive Pulmonary Embolism – A 'Watch-And-Wait' Strategy with Anticoagulation Alone or Advanced Therapy with Thrombolysis

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Abstract - A 65-year old man, presented with syncope and dyspnea. On examination he was tachypneic, hypoxemic, normotensive, with elevated D-dimer and cardiac troponin. ECG showed sinus tachycardia $S_1Q_3T_3$ syndrome and echocardiography revealed right ventricular dysfunction. Urgent computed tomograph angiograms showed bilateral pulmonary embolism. After treatment with intravenous tpA the patient's status improved and echocardiogram showed decreasing of the right ventricular systolic pressure.

The most widely accepted indication for thrombolic therapy is proven pulmonary embolism with cardiogenic shock; therapy is also frequently considered when a patient presents with systemic hypotension without shock. The use of thrombolysis in submassive embolism – that is pulmonary embolism causing right ventricular (RV) dilatation and hypokinesis with systemic hypotension – is debated.[1]

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The most widely accepted indication for thrombolic therapy is proven pulmonary embolism with cardiogenic shock; therapy is also frequently considered when a patient presents with systemic hypotension without shock. The use of thrombolysis in submassive embolism – that is pulmonary embolism causing right ventricular (RV) dilatation and hypokinesis with systemic hypotension – is debated.[1]

The purpose of the study was to demonstrate a case of submassive pulmonary embolism who had an excellent clinical electrocardiographic and echocardiographic response to fibrinolysis.

Keywords : submassive pulmonary embolism, thrombolysis, advanced therapy.

I. Case Report

A 65-year-old man, with a history of recent hospitalization for orthopedic surgery, presented with exertional syncope and progressively worsening dyspnea. On examination he was tachypneic (28 breaths per minute), hypoxemic (oxygen saturation 85 on room air), with heart rate of 115 beats per minute and blood pressure of 130/75 mmHg. The neck vein was distended, there was no heart murmur and the lungs were clear on auscultation. Laboratory evaluation was remarkable for D-dimer 2110 ng/ml (normal < 234 ng/ml) and a cardiac troponin level of 1,7 ng/ml (normal < 0,06 ng/ml). The electrocardiogram (ECG) revealed sinus tachycardia with a rate of 115 per minute, a deep S wave in lead I, a Q wave and inverted T wave in lead III ($S_1Q_3T_3$ syndrome) and a subtle ST elevation in leads aVR and V_1 [Fig. 1]. Transthoracic echocardiography revealed right ventricular dilatation and hypokinesis with moderate tricuspid regurgitation and an estimated right ventricular systolic pressure of 65 mmHg. Doppler studies of the legs showed bilateral proximal deep venous thrombosis, making the diagnosis of pulmonary embolism likely. Urgent contrast-enhanced computed tomograph angiograms [Fig.2] showed bilateral pulmonary embolism. After screening for contraindication to fibrinolysis was performed, the decision was made to proceed with thrombolysis into low risk of bleeding and increased risk of death. 100 mg of tissue plasminogen activator (tpA) was administered over a 2-hour period, and after completion of the fibrinolysis, unfractioned heparin was started without a bolus as a "bridge" to anticoagulation with warfarin. After treatment with intravenous tpA, the patient's respiratory status dramatically improved over a period of several hours, the S wave in lead I and the ST elevation in leads aVR and V_1 on the ECG disappeared and the heart rate slowed down to 78 beats per minute [Fig. 3]. Repeated echocardiography showed that the right ventricular systolic pressure decreased to 35 mmHg. On follow-up, 4 weeks later, the patient's condition was good and the echocardiogram documented normal right ventricular size and function.

II. Discussion

Submassive pulmonary embolism (PE) defines patients who appear hemodynamically stable on admission, but have evidence of right ventricular (RV) dysfunction. This group of patients can be identified by the presence of RV dysfunction detected on physical examination, cardiac biomarkers, ECG, echocardiography and chest CT.

The decision to administer a fibrinolytic agent in addition to heparin anticoagulation requires individualized assessment of the balance of benefits versus risk. Potential benefits include more rapid resolution of symptoms, stabilization of respiratory and cardiovascular function without need for mechanical ventilation or vasopressor support, reduction of RV damage, improved exercise tolerance, prevention of PE recurrence and increased probability of survival. Potential harm includes disabling of fatal haemorrhage, including intracerebral haemorrhage and increased risk of minor haemorrhage, resulting in prolongation of

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hospitalisation and need for blood product replacement. Fibrinolysis is most successful when administered within several days of acute PE. Although the efficacy of fibrinolysis is inversely proportional to the duration of symptoms, effective thrombolysis can be observed up to two weeks after an acute event.

Thrombolysis is indicated in the case of patients with PE who have arterial hypotension or are in shock. In contrast, the benefits of thrombolysis normotensive patients with acute PE are less well established. Results from randomised trials suggested that selected patients with evidence of RV dysfunction and a low risk of bleeding may benefit from early fibrinolysis.[2,3] The decision to select thrombolysis for submassive PE or to maintain anticoagulation alone must be individualized because of paucity of trials to help guide management. On the other hand, some authors conclude that there is no scientific support for thrombolytic therapy in this case.[4] In our patient the dilemma was whether the normotensive patient with RV dysfunction, as detected on echocardiogram and CT scan, and with evidence of myocardial injury, as indicated by a positive troponin test, may benefit from early thrombolytic treatment.

After completion of fibrinolysis, the patient's respiratory status and gas-exchange derangements improved over a period of several hours. The ECG returned to normal and the echocardiogram revealed decreasing of the RV systolic pressure.

III. Conclusion

The normotensive patients with acute PE and evidence of RV dysfunction may benefit from early thrombolysis.

IV. Figure Legends

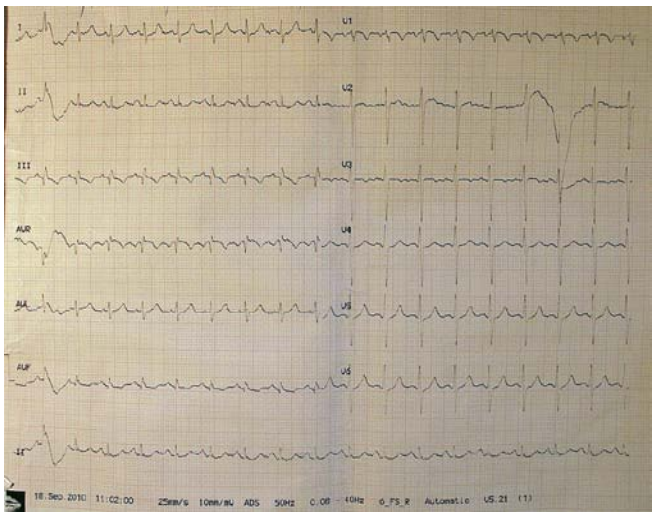

Figure 1 : The ECG on admission shows sinus tachycardia with a rate of 115, a deep S wave in lead I, a Q wave and inverted T wave in lead III ($S_1Q_3T_3$ syndrome) and a subtle ST elevation in leads aVR and V_1

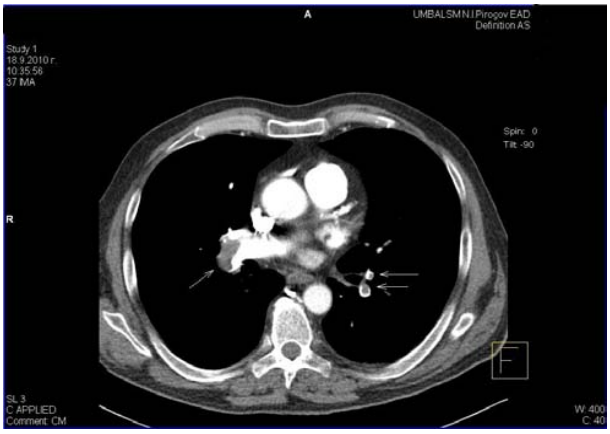

Figure 2 : Multidetector spiral computer tomogram showing bilateral defects (arrows) in the pulmonary artery

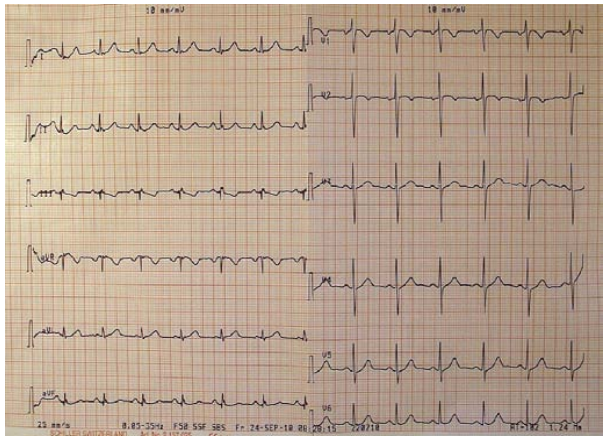

Figure 3 : The ECG performed after completion of fibrinolysis revealed disappearance of S wave in lead I and the ST elevation in leads aVR, V_1 and slowing down of the heart rate to 78

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